


Influence of Secretor Status on the Glucose-6-Phosphate Dehydrogenase Activity

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Abstract:

Background: Glucose 6-phosphate dehydrogenase (G6PD) deficiency is the commonest human enzymopathy. It is particularly common in populations living in malaria-endemic areas, affecting more than 400 million people worldwide. **Objective:** The aim of the present study was to investigate the association of secretor status with G6PD activity. **Materials and Methods:** This was a cross sectional study of 120 serially recruited apparently healthy males. Secretor status of subjects were determined by the hemagglutination test while the G6PD activity was determined by the enzymatic spectrophotometric quantitation assay. Data was analyzed using the Statistical Package for Social Sciences version 23. $P < 0.05$ was considered significant. **Result:** 97% of the secretors showed normal G6PD activity while 5% showed deficient G6PD activity. 22.2% of the non-secretors showed normal G6PD activity while 77.7% showed deficient G6PD activity. **Conclusion:** This finding suggests that non-secretion of ABH substance maybe a risk factor for reduced G6PD activity or deficiency.

Keywords: G6PD, Secretor status, Enugu.

Introduction

Glucose-6-phosphate dehydrogenase (G6PD) is a rate limiting enzyme in the glycolytic pathway which is involved in the production of reduced nicotinamide dinucleotide phosphate (NADPH) that functions to combat oxidative stress and injury to red blood cells thereby protecting them from hemolysis [1]. It's deficiency is the most common human enzymopathy known globally and in sub-Saharan African, prevalence rate as high as 32.5% has been reported [2]. Secretors denote individuals whose blood group antigens are expressed in their body fluid (saliva, urine, semen, serum, colostrum) while non-secretors are individuals whose blood group antigens are not expressed in their body fluids. The secretor genotype is denoted as SeSe/Sese while the non-secretor genotype is denoted as sese [3,4]. Studies have demonstrated

the association of secretor status with different diseases in many populations but there is currently a paucity of data on the association of secretor status with G6PD activity for the Nigerian population [5]. The aim of this study was to investigate the association of secretor status with G6PD activity among individuals in Enugu, Nigeria.

Materials and Methods

Study Setting

The study was carried out in the Enugu State University of Science and Technology (ESUT) Teaching Hospital, Enugu, Nigeria. The ESUT Teaching Hospital is the major tertiary health facility for the State and is located at the center of the capital city also known as Enugu for easy accessibility to residents. Enugu State is made up of three senatorial zones namely Enugu East, Enugu West and North. The senatorial zones are divided into seventeen Local Government Areas comprising 450 communities. The State derived its name from the name of its capital and largest city, Enugu. It has an area of 7,161km² with a population of 3,267,837 lies between longitudes 6°30'E and latitudes 5°15'N and 7°15'E. It is bordered by Abia State and Imo States to the South, Ebonyi to the East, Benue and Kogi States to the North and Anambra to the West. It comprises mainly the Igbo speaking tribe of South Eastern Nigeria, about 50% of which lives in the rural areas [6].

Study Design

This was a across sectional study involving 120 apparently healthy males aged 40 years and above recruited by convenient sampling within Enugu State University Teaching Hospital between April and December, 2023.

Collection of Urine Sample

After proper rinsing of the mouth of each participant with distilled water, a clean rubber band was given to the participants to chew to increase salivation. After discarding the first few drops, about 3mls of saliva was collected into a sterile plain container for the determination of secretor status.

Collection of Blood Sample

Three (3) mls of blood was collected into ethylene amine tetra acetic acid bottles from subjects using standard venipuncture technique for the determination of G6PD activity.

Determination of G6PD Activity

All blood samples were screened for G6PD activity based on the spectrophotometric quantitation of G6PD level using Randox diagnostic kit (Ardmore Diamond Road, Crumlin, Country Antrim, United Kingdom) with decreased activity or G6PD deficiency taken as any level of enzyme activity less than 6.97 iu/g Hemoglobin (reference range 6.92-20.5 iu/g Hemoglobin). The Randox G6PD kit works on the

principle of the ability of the enzyme to reduce NADP to NAPH with the rate of reduction of NADP⁺ measured at 340nm.

Determination of the Secretor Status

Three milliliters (3mls) of saliva was transferred into a test tube and placed in a boiling water bath for 10 minutes to denature salivary enzymes. It was then cooled and centrifuged at 10,000 RPM for 5 minutes; the supernatant was harvested, equal volume of it was placed into three labelled tubes A, B and H. Equal volume of diluted Anti-A, Anti-B, and Anti-H, were added to the appropriate tubes; Antisera-A into tube A, Antisera-B into B, Antisera-H into H tubes respectively. Control were included to ascertain the antisera. Each tube was mixed and incubated at room temperature for about 10 minutes. A drop of standard red cells A, B and H was added into the corresponding tubes A, B and H. Each content of the tubes was mixed and incubated further for 10 minutes at room temperature. Reaction or agglutination were observed. Control tubes was also observed for agglutination to confirm potency of antisera. Absence of corresponding A, B and/or H soluble antigen (substance). Agglutination in any test sample tube indicated absence of soluble secretor antigen A, B and/or H.

Sample Size

The required minimum sample size was determined using the Lesly kish formula for estimating minimum sample size in health studies [7].

$$n = \frac{Z^2Pq}{d^2}$$

where

n = desired sample size

p = prevalence of G6PD deficiency in Nigeria population, 20% = 0.20 [8];

d = desired precision limit at 5% = 0.05

Z = confidence interval set at 0.05 = 1.96.

Substituting

$$\begin{aligned} n &= \frac{1.96^2 \times 0.20 \times 0.20}{0.0025} \\ &= 61 \end{aligned}$$

Inclusion Criteria

Individuals who have normal full blood count results at time of study.

Individuals with no underlying hemoglobinopathy.

Individuals who are not under specific medications such as α -methyl dopa (antihypertensive) which could exacerbate red cell lysis.

Exclusion Criteria

Individuals who have abnormal full blood count result at time of study.

Individuals with an underlying hemoglobinopathy.

Individuals who were under some medications such as α -methyl dopa.

Ethical Considerations

The ethical clearance for the study was obtained from the Ethics Committee of the Enugu State University of Science and Technology Teaching Hospital, Enugu with reference number: NP/C-MAC/RA/035/Vol 4/38. Informed consent was obtained from all subjects before recruitment for the study.

Data Analysis

Data was analyzed using IBM Statistical Package for Social Sciences (SPSS) for windows version 23, Armok, NY, USA. This was presented with descriptive statistics as frequency and percentages.

Result

An evaluation of the secretor status of the subjects revealed that 85% of the subjects are secretors while 15% are non-secretor Table 1 while the evaluation of the G6PD status of the subjects revealed that 95% of the subjects have normal G6PD activity and 25% have deficient G6PD activity Table 2. Among the subjects who are secretors, 97% showed normal G6PD activity while 5% showed deficient G6PD activity Table 3 while among the subjects who are non-secretors 22.2% showed normal G6PD activity and 77.7% showed deficient G6PD activity Table 4.

Table 1. Secretor Status of the Subjects

Secretor status	Frequency	Percentage
Secretors	102	85
Non secretors	18	15
TOTAL	120	100

Table 2. G6PD Status of the Subjects

G6PD Status	G6PD Activity (iu/gHb)	Frequency	Percentage
Normal	10.51 \pm 2.7	95	79.2
Deficient	4.36 \pm 2.3	25	20.8

Table 3. Prevalence of G6PD deficiency among the subjects who are secretors

G6PD Status	G6PD Activity (iu/gHb)	Frequency	Percentage
Normal	9.72 \pm 2.82	4	22.2
Deficient	3.12 \pm 1.94	14	77.7

Table 4. Prevalence of G6PD deficiency among the subjects who are non-secretors

G6PD Status	G6PD Activity (iu/gHb)	Frequency	Percentage
Normal	11.30 \pm 2.82	4	22.2
Deficient	3.12 \pm 1.94	14	77.7

Discussion

Given that the distribution of several diseases has been linked to the secretor status of individuals, we investigated the association between G6PD activity and secretor status among apparently healthy individuals. The prevalence of G6PD deficiency among the subjects who are secretors revealed that 95% have normal G6PD activity while only 4.9% are G6PD deficient. On the other hand, the prevalence of G6PD deficiency among the subjects who are non-secretors revealed that 4% have normal G6PD activity while 14% are G6PD deficient. This finding is similar to the reports on the potential health disadvantage of non-secretors compared to secretors as a good number of both communicable and non-communicable diseases has been associated with the non ABH secretory status in different populations [9-11]. Individuals with reduced G6PD activity are prone to oxidative stress due to high rate of reactive oxygen species generated during normal metabolism following the ingestion of certain foods or drugs. Considering the high prevalence of G6PD deficiency among individuals in the Sub-Saharan Africa, it is pertinent that we identify the various determinants of the enzyme activity for this population which is a gap that the present study seeks to address. A limitation to the present study is the small sample size and the use of a single center, therefore further studies involving larger sample size, subjects from a diverse community and multi centers are recommended to support the present finding.

Conclusion

It can be concluded based on the present data that individuals who are non-secretors are susceptible to reduced G6PD activity or deficiency of the enzyme compared to secretors.

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